

=> D HIS

(FILE 'HOME' ENTERED AT 17:22:44 ON 20 JUN 2006)

FILE 'STNGUIDE' ENTERED AT 17:22:56 ON 20 JUN 2006

FILE 'MEDLINE' ENTERED AT 17:23:07 ON 20 JUN 2006

L1 24868 S GH OR GROWTH HORMONE! OR SOMATOTROPIN! OR SOMATOTROPHIN!  
L2 227469 S HYPOXI! OR ISCHEMI!  
L3 144 S L1 AND L2  
L4 2955 S L1 AND NEURO?  
L5 2933 S L4 NOT L3  
L6 72 S L5 AND (RESCUE? OR PROTECT? OR PROPHYL?)  
L7 532 S L5 AND (CNS OR BRAIN)  
L8 518 S L7 NOT L6

=>

5,958, 933 9/28/99  
filed 6/6/95  
column 142, line 13-15

WO 2001-JP/04/16  
JP 2000-367/83

#256 6/21, 4/6 - 1997  
#237 - date?  
6469024 5/10/01  
5/11/00 provisional  
MS + GH reaction

~~#236 6,352,982~~  
~~WO 98/42882~~  
~~10/29/98~~

=> D BIB AB 1-9

L12 ANSWER 1 OF 9 MEDLINE on STN

AN 2005240737 MEDLINE

DN PubMed ID: 15879686

TI Treatment in animal models.

AU Guan J; Bennet L; Gluckman P D; Gunn A J

CS The Liggins Institute, Faculty of Medicine and Health Sciences, The

University of Auckland, Auckland, New Zealand..

j.guan@auckland.ac.nz

NC R01 HD-32752 (NICHHD)

SO Endocrine development, (2005) Vol. 9, pp. 31-43. Ref: 27

Journal code: 101138956. ISSN: 1421-7082.

CY Switzerland

DT Journal; Article; (JOURNAL ARTICLE)

General Review; (REVIEW)

LA English

FS Priority Journals

EM 200507

ED Entered STN: 10 May 2005

Last Updated on STN: 9 Jul 2005

Entered Medline: 8 Jul 2005

AB It is now well established that neurons and other cell types may die many hours or even days after hypoxic-ischemic injury due to activation of programmed cell death (apoptotic) pathways. The potent anti-apoptotic factor IGF-1 and its binding proteins and receptors are intensely induced within damaged brain regions following brain injury suggesting a possible role for IGF-1 in endogenous brain recovery. Exogenous administration of IGF-1 within a few hours after brain injury has now been shown to be protective in both grey and white matter, and leads to improved long-term neurological function. The limited window of opportunity for treatment with IGF-1 can be extended by spontaneous mild post-hypoxic hypothermia, probably due to delayed evolution of apoptotic processes. The efficacy of IGF-1 is specific to particular cellular phenotypes and brain regions, and its neuroprotective effects are mediated by IGF-1 receptors and binding proteins. Intriguingly its naturally cleaved N-terminal tripeptide ( \*\*\*glycine\*\*\* - \*\*\*proline\*\*\* - \*\*\*glutamate\*\*\* , \*\*\*GPE\*\*\* ) has been demonstrated to be neuroprotective after both central and peripheral administration. Peripheral administration of \*\*\*GPE\*\*\* also prevents the loss of dopamine neurons and improves long-term functional recovery following 6-OHDA lesion. However, \*\*\*GPE\*\*\* is unlikely to contribute significantly to the direct effects of IGF-1.

L12 ANSWER 2 OF 9 MEDLINE on STN

AN 2005123223 MEDLINE

DN PubMed ID: 15752541

TI Central penetration and stability of N-terminal tripeptide of insulin-like

growth factor-I, \*\*\*glycine\*\*\* - \*\*\*proline\*\*\* - \*\*\*glutamate\*\*\* in adult rat.

AU Baker A M; Batchelor D C; Thomas G B; Wen J Y; Rafiee M; Lin H; Guan J

CS Liggins Institute, Faculty of Medical and Health Science, The University

of Auckland, Private Bag 92019, Auckland, New Zealand.

SO Neuropeptides, (2005 Apr) Vol. 39, No. 2, pp. 81-7.

Electronic

Publication: 2005-01-28.

Journal code: 8103156. ISSN: 0143-4179.

CY Scotland: United Kingdom

DT Journal; Article; (JOURNAL ARTICLE)

LA English

FS Priority Journals

EM 200505

ED Entered STN: 9 Mar 2005

Last Updated on STN: 6 May 2005

Entered Medline: 5 May 2005

AB Insulin-like growth factor-I is a neurotrophic factor and can prevent

neurons from ischemic brain injury. However, the large molecular weight

and metabolic effects can be problematic in its central delivery.

\*\*\*Glycine\*\*\* - \*\*\*proline\*\*\* - \*\*\*glutamate\*\*\* ( \*\*\*GPE\*\*\* ) is

the N-terminal tripeptide of insulin-like growth factor-I, which is

naturally cleaved in the plasma and brain tissues.

\*\*\*GPE\*\*\* reduces

neuronal loss from hypoxic-ischemic brain injury following central

administration. Central penetration and the stability of \*\*\*GPE\*\*\* in

the plasma and central nervous system were examined in rats using

radioimmunoassay and HPLC. \*\*\*GPE\*\*\* was rapidly metabolised in the

plasma (8 min) after intraperitoneal administration. Despite having a

short half-life in plasma, \*\*\*GPE\*\*\* was detected in the cerebrospinal

fluid up to 40 min after intraperitoneal administration. With present of

peptidase inhibitors, \*\*\*GPE\*\*\* existed in the brain tissue up to 3 h

after intracerebroventricular administration, suggesting a role for

peptolysis in its stability. The endopeptidase inhibitors 4-(2-aminoethyl) benzenesulfonyl fluoride hydrochloride (AEBSF) reduced

\*\*\*GPE\*\*\* metabolism in the brain tissue while acid peptidase inhibitor

pepstatin-A decreased \*\*\*GPE\*\*\* metabolism in the plasma. \*\*\*GPE\*\*\*

reduced neuronal loss in the CA1-2 sub-region of the hippocampus given

(intraperitoneally) after 30 min of hypoxic-ischemic injury in adult rats,

further suggested the effectiveness of \*\*\*GPE\*\*\* central uptake.

These results indicated that \*\*\*GPE\*\*\* crosses the blood-CSF and the

functional CSF-brain barriers. The longer half-life of \*\*\*GPE\*\*\* in

the CNS may be due to its unique enzymatic stability.

L12 ANSWER 3 OF 9 MEDLINE on STN

AN 2004556945 MEDLINE  
 DN PubMed ID: 15527823  
 TI Neuroprotective effects of the N-terminal tripeptide of insulin-like growth factor-1, \*\*\*glycine\*\*\* - \*\*\*proline\*\*\* - \*\*\*glutamate\*\*\* (\*\*\*GPE\*\*\* ) following intravenous infusion in hypoxic-ischemic adult rats.  
 AU Guan J; Thomas G B; Lin H; Mathai S; Bachelor D C; George S; Gluckman P D  
 CS The Liggins Institute, Faculty of Medical and Health Sciences, University of Auckland, Private Bag 92019, 2-6 Park Avenue, Grafton, Auckland, New Zealand.. j.guan@auckland.ac.nz  
 SO Neuropharmacology, (2004 Nov) Vol. 47, No. 6, pp. 892-903.  
 Journal code: 0236217. ISSN: 0028-3908.  
 CY England: United Kingdom  
 DT Journal; Article; (JOURNAL ARTICLE)  
 LA English  
 FS Priority Journals  
 EM 200501  
 ED Entered STN: 6 Nov 2004  
 Last Updated on STN: 14 Jan 2005  
 Entered Medline: 13 Jan 2005  
 AB The N-terminal tripeptide of insulin-like growth factor-1, \*\*\*GPE\*\*\* is neuroprotective when given intracerebroventricularly 2 h after hypoxic-ischemic (HI) brain injury in rats. We have now examined whether \*\*\*GPE\*\*\* can cross the blood-brain barrier and exert neuroprotective actions following intravenous administration. Following a single bolus intravenous injection, \*\*\*GPE\*\*\* was rapidly metabolized and cleared from the circulation. The short half-life (<2 min) in blood was subsequently associated with modest and inconsistent neuroprotection. In contrast, potent neuroprotection of \*\*\*GPE\*\*\* was consistently observed in all brain regions examined following 4 h intravenous infusion (12 mg/kg). The neuroprotective effects of \*\*\*GPE\*\*\* after infusion showed a broad effective dose range (1.2-120 mg/kg) and an extended window of treatment to 7-11 h after injury. The central penetration of \*\*\*GPE\*\*\* after intravenous infusion was injury-dependent. \*\*\*GPE\*\*\* also improved long-term somatofunction with a comparable neuronal outcome. \*\*\*GPE\*\*\* reduced both caspase-3-dependent and -independent apoptosis in the hippocampus. Treatment with \*\*\*GPE\*\*\* also inhibited microglial proliferation and prevented the injury-induced loss of astrocytes. In conclusion, the neuroprotective actions of \*\*\*GPE\*\*\* infusion were global, robust and displayed a broad effective dose range and treatment window. \*\*\*GPE\*\*\*'s activity included the prevention of neuronal

apoptosis, promotion of astrocyte survival and inhibition of microglial proliferation. With injury specific central penetration, \*\*\*GPE\*\*\* has considerable promise as a systemic neuroprotective treatment after acute encephalopathies.

L12 ANSWER 4 OF 9 MEDLINE on STN  
 AN 2003576269 MEDLINE  
 DN PubMed ID: 14656520  
 TI Pharmacokinetics of \*\*\*glycine\*\*\* - \*\*\*proline\*\*\* - \*\*\*glutamate\*\*\*, the N-terminal tripeptide of insulin-like growth factor-1, in rats.  
 AU Batchelor D C; Lin H; Wen J-Y; Keven C; Van Zijl P L; Breier B H; Gluckman P D; Thomas G B  
 CS NeuronZ Ltd, PO Box 9923, Newmarket, Auckland 1031, New Zealand.. david.batchelor@neuronz.com  
 SO Analytical biochemistry, (2003 Dec 15) Vol. 323, No. 2, pp. 156-63.  
 Journal code: 0370535. ISSN: 0003-2697.  
 CY United States  
 DT Journal; Article; (JOURNAL ARTICLE)  
 LA English  
 FS Priority Journals  
 EM 200408  
 ED Entered STN: 16 Dec 2003  
 Last Updated on STN: 1 Sep 2004  
 Entered Medline: 31 Aug 2004  
 AB \*\*\*Glycine\*\*\* - \*\*\*proline\*\*\* - \*\*\*glutamate\*\*\* (\*\*\*GPE\*\*\* ) is the N-terminal tripeptide of insulin-like growth factor-1 and has been shown to be neuroprotective following ischemia-induced brain injury. The pharmacokinetics of \*\*\*GPE\*\*\* were studied in adult rats since \*\*\*GPE\*\*\* is a candidate for use in neuroprotection therapies. To measure plasma concentrations of \*\*\*GPE\*\*\* a novel radioimmunoassay was developed whereby \*\*\*GPE\*\*\* was initially derivatized with Bolton and Hunter reagent before use in a standard homologous assay against the Bolton and Hunter iodinated form. The derivatized \*\*\*GPE\*\*\* radioimmunoassay showed a 83% recovery of unlabeled \*\*\*GPE\*\*\* and complete parallel displacement with rat plasma. The simplicity and speed of the assay described here indicate an exciting new use for a previously described technology. The pharmacokinetic studies were conducted in adult rats using a single bolus intravenous injection of \*\*\*GPE\*\*\* at 30 or 100 mg/kg and showed that \*\*\*GPE\*\*\* was rapidly cleared from the circulation. In addition, evidence of the route of the metabolic degradation of \*\*\*GPE\*\*\* is presented. The findings presented here are the first description of the pharmacokinetics of \*\*\*GPE\*\*\* and suggest that, because of its very short half-life in plasma, continuous

intravenous infusion of \*\*\*GPE\*\*\* may be the preferred route of administration for use in future neuroprotection therapies.

L12 ANSWER 5 OF 9 MEDLINE on STN

AN 2002046495 MEDLINE

DN PubMed ID: 11730700

TI Neuroprotective effects of the N-terminal tripeptide of IGF-1,

\*\*\*glycine\*\*\* - \*\*\*proline\*\*\* - \*\*\*glutamate\*\*\*, in the immature rat

brain after hypoxic-ischemic injury.

AU Sizonenko S V; Sirimanne E S; Williams C E; Gluckman P D

CS Liggins Institute, Faculty of Medicine and Health Science, The University

of Auckland, Private Bag 92019, Auckland, New Zealand..

stephane.sizonenko@medicine.unige.ch

SO Brain research, (2001 Dec 13) Vol. 922, No. 1, pp. 42-50. Journal code: 0045503. ISSN: 0006-8993.

CY Netherlands

DT Journal; Article; (JOURNAL ARTICLE)

LA English

FS Priority Journals

EM 200202

ED Entered STN: 24 Jan 2002

Last Updated on STN: 20 Feb 2002

Entered Medline: 19 Feb 2002

AB Insulin growth factor 1 (IGF-1) has an important role in brain development

and is strongly expressed during recovery after a hypoxic-ischemic injury.

Some of its central actions could be mediated through the N-terminal

tripeptide fragment of IGF-1: Gly-Pro-Glu ( \*\*\*GPE\*\*\* ).

The

neuroprotective properties of \*\*\*GPE\*\*\* given after a moderate injury

in the developing rat brain were evaluated and the binding sites of [(3)H]

\*\*\*GPE\*\*\* characterised by autoradiography. After right unilateral

injury, \*\*\*GPE\*\*\* or vehicle (V) was injected in the right lateral

ventricle (i.c.v.) or in the peritoneal cavity (i.p.) of 21-day-old rats.

The percentage of surviving neurons in CA1-2 of the hippocampus was higher

in the animals treated with 30 microg of \*\*\*GPE\*\*\* i.c.v. (V:

7.7+/-4.9%, \*\*\*GPE\*\*\* : 26.4+/-7.5%, P=0.02) and 300 microg i.p. (V:

30.2+/-9.1%, \*\*\*GPE\*\*\* : 68.8+/-10.6%, P=0.02) than in animals

receiving vehicle. I.p. injection of 300 microg of \*\*\*GPE\*\*\* (V:

78.4+/-7.5%, \*\*\*GPE\*\*\* : 88.4+/-3.2%, P=0.04) was also neuroprotective

in the lateral cortex. I.c.v. injection of [(3)H] \*\*\*GPE\*\*\* suggested

binding to glial cells in the white matter tracts, the cortex and striatum .

as opposed to neurons. Although the precise mode of action of \*\*\*GPE\*\*\*

is unknown, this study suggests that local administration of \*\*\*GPE\*\*\*

is neuroprotective after brain HI injury via glial cells. In addition,

systemic administration of \*\*\*GPE\*\*\* showed a more widespread

neuroprotective effect. \*\*\*GPE\*\*\* may represent a complementary pathway for central and systemic IGF-1's antiapoptotic effects.

L12 ANSWER 6 OF 9 MEDLINE on STN

AN 2001177905 MEDLINE

DN PubMed ID: 11202225

TI A role for the somatotrophic axis in neural development, injury and

disease.

AU Scheepens A; Williams C E; Breier B H; Guan J; Gluckman P D

CS Research Centre for Developmental Medicine and Biology, Faculty of Medical

and Health Science, University of Auckland, New Zealand..

a.scheepens@auckland.ac.nz

SO Journal of pediatric endocrinology & metabolism : JPEM, (2000) Vol. 13

Suppl 6, pp. 1483-91. Ref: 78

Journal code: 9508900. ISSN: 0334-018X.

CY England: United Kingdom

DT Journal; Article; (JOURNAL ARTICLE)

General Review; (REVIEW)

LA English

FS Priority Journals

EM 200103

ED Entered STN: 4 Apr 2001

Last Updated on STN: 4 Apr 2001

Entered Medline: 29 Mar 2001

AB This review article discusses the roles of the somatotrophic axis in the

growth and development of the normal central nervous system (CNS) and

during recovery from brain injury. Classically, the actions of

pituitary-derived growth hormone (GH) have been reported to be primarily

mediated via the induction of hepatic insulin-like growth factor-I

(IGF-I). GH receptors (GHRs), however, have now been identified in many

body tissues and shown to have both endocrine and local actions, some of

which are IGF-I independent. Within the brain, GHRs are widely located

across a range of cellular phenotypes, yet little is known regarding their

function or endogenous ligand. It is now becoming accepted that GH, like

IGF-I, is integrally involved in the growth and development of the normal

CNS. Following brain injury, IGF-I mRNA is induced, primarily within

reactive microglia. The resultant IGF-I protein appears to have a dual

role, first as an endogenous neurotropic and anti-apoptotic agent acting

directly on stressed cells, and second as a prohormone for generation of

the N-terminal tripeptide of IGF-I, \*\*\*glycine\*\*\* - \*\*\*proline\*\*\* -

\*\*\*glutamate\*\*\* ( \*\*\*GPE\*\*\* ), and the resulting des-N-(1-3)-IGF-I,

both of which have specific neuroprotective properties. Our work on

deciphering the upstream regulators of injury-induced IGF-I has revealed

that a GH-like substance is strongly upregulated after brain injury and

specifically associated with stressed neurons and glia. Subsequent to this finding, GH administered centrally 2 hours after a hypoxic-ischemic brain injury in juvenile rats was found to provide significant neuroprotection, interestingly, in a spatiotemporal pattern distinct from the neuroprotection offered by IGF-I. The implications of these findings in regard to the growth, development and injury response of the CNS are discussed.

L12 ANSWER 7 OF 9 MEDLINE on STN

AN 2000185378 MEDLINE

DN PubMed ID: 10719076

TI N-terminal tripeptide of IGF-1 ( \*\*\*GPE\*\*\* ) prevents the loss of TH

positive neurons after 6-OHDA induced nigral lesion in rats. AU Guan J; Krishnamurthi R; Waldvogel H J; Faull R L; Clark R; Gluckman P

CS Research Center for Developmental Medicine, School of Medicine, University of Auckland, Private Bag 92019, Auckland, New Zealand.. j.guan@auckland.ac.nz

SO Brain research, (2000 Mar 24) Vol. 859, No. 2, pp. 286-92.

Journal code: 0045503. ISSN: 0006-8993.

CY Netherlands

DT Journal; Article; (JOURNAL ARTICLE)

LA English

FS Priority Journals

EM 200005

ED Entered STN: 18 May 2000

Last Updated on STN: 18 May 2000

Entered Medline: 9 May 2000

AB The effect of the N-terminal tripeptide of insulin-like growth factor (IGF)-1, \*\*\*glycine\*\*\* - \*\*\*proline\*\*\* - \*\*\*glutamate\*\*\* ( \*\*\*GPE\*\*\* ), as a neuroprotective agent for nigro-striatal dopaminergic

neurons was examined in the present study using a rat model of Parkinson's disease. A unilateral nigro-striatal lesion was induced in rats by injecting 6-hydroxydopamine (6-OHDA) into the right medial forebrain bundle (MFB). \*\*\*GPE\*\*\* (3 microgram) or its vehicle was administered intracerebroventricularly (i.c.v.) 2 h after the 6-OHDA lesion.

Tyrosine-hydroxylase (TH) immunohistochemistry in the substantia nigra compacta (SNc) and the striatum were examined 2 weeks after the lesion.

Following 6-OHDA injection, the number of TH immunopositive neurons in the ipsilateral SNc was reduced. The density of TH immunostaining was also reduced in the ipsilateral SNc and the striatum. Treatment with a single dose of \*\*\*GPE\*\*\* (n=9) significantly prevented the loss of TH

immunopositive neurons (p<0.001) and restored the TH immunoreactivity in both the SNc and the striatum compared with the vehicle control group (n=9, p<0.001). The results suggest that \*\*\*GPE\*\*\* showed promise as a potential treatment for Parkinson's disease.

L12 ANSWER 8 OF 9 MEDLINE on STN

AN 1999417601 MEDLINE

DN PubMed ID: 10486177

TI The IGF-I amino-terminal tripeptide \*\*\*glycine\*\*\* - \*\*\*proline\*\*\* -

\*\*\*glutamate\*\*\* ( \*\*\*GPE\*\*\* ) is neuroprotective to striatum in the quinolinic acid lesion animal model of Huntington's disease. AU Alexi T; Hughes P E; van Roon-Mom W M; Faull R L; Williams C E; Clark R G;

Gluckman P D CS School of Medicine, University of Auckland, Auckland, New Zealand..

t.alex@auckland.ac.nz

SO Experimental neurology, (1999 Sep) Vol. 159, No. 1, pp. 84-97.

Journal code: 0370712. ISSN: 0014-4886.

CY United States

DT Journal; Article; (JOURNAL ARTICLE)

LA English

FS Priority Journals

EM 199910

ED Entered STN: 14 Oct 1999

Last Updated on STN: 3 Mar 2000

Entered Medline: 7 Oct 1999

AB Huntington's disease is an incurable genetic neurological disorder

characterized by the relatively selective degeneration of the striatum.

Lesioning of the striatum in rodents using the excitatory amino acid

agonist, quinolinic acid (QA), effectively mimics the human neuropathology

seen in Huntington's disease. Using this animal model of Huntington's

disease, we investigated the ability of the insulin-like growth factor-I

(IGF-I) amino-terminal tripeptide \*\*\*glycine\*\*\* - \*\*\*proline\*\*\* -

\*\*\*glutamate\*\*\* ( \*\*\*GPE\*\*\* ) to protect striatal neurons from

degeneration. Adult rats received a single unilateral intrastratial

injection of QA (100 nmol) and then daily injection of either vehicle or

\*\*\*GPE\*\*\* (0.3 microgram/microliter/day) into the striatum for 7 days.

QA at this dose resulted in a partial lesioning of the striatum after 7

days to approximately 50% of cells of unlesioned levels in vehicle-treated

animals. The major striatal neuronal phenotype,

GABAergic projection

neurons, were identified by immunocytochemical labeling of either

glutamate decarboxylase 67 (GAD(67)) or the calcium binding protein

calbindin in alternate sections. Treatment with \*\*\*GPE\*\*\* for 7 days

reversed the loss in projection neurons when assessed by counts of

calbindin-stained cells; however, these rescued cells did not regain

immunologically detectable levels of GAD(67). \*\*\*GPE\*\*\* also

significantly reversed the phenotypic degeneration of cholinergic

interneurons identified by immunolabeling for choline acetyltransferase

(ChAT) and NADPH diaphorase interneurons identified histochemically.  
 \*\*\*GPE\*\*\* treatment failed to rescue the calcium binding protein  
 interneuron populations of parvalbumin and calretinin neurons. These  
 findings reveal that exogenous administration of  
 \*\*\*GPE\*\*\* selectively  
 prevents excitotoxin induced phenotypic degeneration of striatal  
 projection neurons and cholinergic and NADPH diaphorase interneurons in an  
 animal model of Huntington's disease.  
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L12 ANSWER 9 OF 9 MEDLINE on STN  
 AN 1999191943 MEDLINE  
 DN PubMed ID: 10094155  
 TI Neuroprotective effects of Gly-Pro-Glu, the N-terminal tripeptide of  
 IGF-1, in the hippocampus in vitro.  
 AU Saura J; Curatolo L; Williams C E; Gatti S; Benatti L; Peeters C; Guan J;  
 Dragunow M; Post C; Faull R L; Gluckman P D; Skinner S J  
 CS Department of Pharmacology, School of Medicine, University of Auckland,  
 New Zealand.  
 SO Neuroreport, (1999 Jan 18) Vol. 10, No. 1, pp. 161-4.  
 Journal code: 9100935. ISSN: 0959-4965.  
 CY ENGLAND: United Kingdom  
 DT Journal; Article; (JOURNAL ARTICLE)  
 LA English  
 FS Priority Journals  
 EM 199905  
 ED Entered STN: 7 Jun 1999  
 Last Updated on STN: 7 Jun 1999  
 Entered Medline: 25 May 1999  
 AB Insulin-like growth factor 1 (IGF-1) plays a critical role in CNS  
 development. IGF-1 can block neuronal apoptosis in vitro and in vivo.  
 IGF-1 is thought to be cleaved into des-N-(1-3)-IGF-1 and an amino  
 terminal \*\*\*glycine\*\*\* - \*\*\*proline\*\*\* - \*\*\*glutamate\*\*\* (\*\*\*GPE\*\*\* tripeptide). Here we report a neuroprotective role for  
 \*\*\*GPE\*\*\* tripeptide, with enhanced survival of the CA1-2 hippocampal  
 neurons following an excitotoxic insult in vitro. Binding and displacement studies suggest uniquely distributed sites of action within  
 the rat including the hippocampal CA1-2, pyriform cortex, amygdala,  
 choroid plexus, blood vessels and to a lesser extent in the cortical  
 regions. A similar pattern of binding was seen in the human. This  
 finding could lead to new strategies to reduce neuronal death after injury  
 and in disease.

=> D HIS

(FILE 'HOME' ENTERED AT 20:42:27 ON 14 JUN 2006)

FILE 'MEDLINE' ENTERED AT 20:42:36 ON 14 JUN 2006

L1 24174 S GH  
 L2 485 S GPE

L3 1 S L1 AND L2  
 L4 52966 S GROWTH HORMONE  
 L5 56694 S L1 OR L4  
 L6 1 S L2 AND L5  
 L7 10115 S SOMATOTROPIN  
 L8 57663 S L5 OR L7  
 L9 1 S L8 AND L2  
 L10 12 S GLYCINE-PROLINE-GLUTAMATE  
 L11 1 S L10 AND L8  
 L12 9 S L10 AND L2

113 51\*

109\* 34\*

106 31\*

105 19

103 11

100 7\*

92

91

90\*

82

73

66

61

55\*